PS07-326

Xanthomonas Type III effector XopD desumoylates tomato transcription factor SlERF4 to suppress ethylene responses and promote pathogen growth

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Manipulation of host protein sumoylation by pathogens has emerged as an important virulence strategy to suppress immunity. The direct link between protein sumoylation and eukaryotic transcription suggests that pathogens might directly modulate the sumoylation state of transcription factors. Here we provide evidence that XopD, a SUMO protease from *Xanthomonas campestris* pathovar *vesicatoria* (Xcv), directly interferes with plant transcription to modulate ethylene (ET) responses during infection. XopD is required to promote Xcv growth in tomato leaves and to suppress disease symptom development. Given that XopD contains two EAR motifs implicated in ET signaling and transcription repression, we hypothesized that XopD may directly regulate ET production and/or signaling. Consistent with this hypothesis, ET gas and biosynthesis mRNAs were significantly higher in Xcv delta*xopD*-infected leaves compared to Xcv-infected leaves. Both ET production and perception were required for tomato immunity and symptom development. Inspection of tomato ERFs expressed in Xcv-infected leaves suggested that SlERF4 is a putative XopD substrate. Virus-induced gene silencing in tomato revealed that *SlERF4* mRNA expression was required for Xcv delta*xopD*-induced ET production and ET-stimulated immunity. XopD was found to colocalize with SlERF4 in subnuclear foci and hydrolyze tomato SUMO1 from K53 of SlERF4 resulting in SlERF4 destabilization. Mutation of K53 to R53 prevented SlERF4 sumoylation, decreased SlERF4 levels, and reduced SlERF4-dependent transcription. We conclude that XopD directly binds and desumoylates SlERF4 to repress ET induced-transcription required for Xcv immunity. This is the first example of a pathogen SUMO protease that targets a host sumoylated transcription factor to suppress defense.

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Dissecting the interaction between *P. syringae* pv. *phaseolicola* and its non-host *A. thaliana* using effectoromics

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MAMP-triggered immunity (MTI) is thought to be a major determinant of non-host resistance (NHR) of Arabidopsis to *P. syringae* pv. *phaseolicola* 1448A (Pph1448A). However, Pph1448A produces effectors that can be potentially recognized and induce effector-triggered immunity (ETI) in Arabidopsis including AvrRps4, HopAS1, and three AvrB homologs: AvrB2, AvrB4-1 and AvrB4-2. To establish the contribution of ETI to the incompatibility between Arabidopsis and Pph1448A, we determined the patterns of effector recognition among different Arabidopsis ecotypes. We used a Tobacco Rattle Virus-based transient expression system to deliver effectors individually and analyzed their ability to induce ETI based on the phenotype of infected plants. Recognition was manifested as symptomless immunity or as extensive necrosis associated with the induction of a hypersensitive response. All three AvrB homologs triggered RPM1/RIN4 and TAO1-mediated defenses in Col-0. In addition, two AvrB4 paralogs triggered RPM1/TAO1-independent defenses in Col-0 due to RPS2 activation. Also HopJ1 triggered defense responses in Col-0 and several other ecotypes. We mapped this response to a ~10cM region in the Arabidopsis genome and using a reverse genetic approach narrowed down the determinant of recognition to a single CC-NB-LRR-encoding gene with no known specificity reported previously. We named this gene *DERK1* for *Determinant of Effector Recognition 1*. We are pyramiding several knockouts of NB-LRR encoding genes to produce lines that are compromised in recognition of effectors from Pph1448A. These will enable us to determine the quantitative contributions of ETI and MTI to NHR of Arabidopsis to Pph1448A.

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Identification and characterization of intracellular effectors Crinklers of the Oomycete *Aphanomyces euteiches*, a root pathogen of legumes

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Aphanomyces euteiches is an oomycete infecting roots of various legumes species as pea, alfalfa and the model legume *Medicago truncatula*. The genus *Aphanomyces* (Saprolegniales) has a particular taxonomic position within oomycetes comprising both animal pathogen and plant pathogen species. cDNA libraries from infectious mycelium revealed the presence of ortholog CRN (Crinkling and Necrosis) genes, initially identified in *Phytophthora infestans*. CRN proteins of *Phythophthora sp* are coded by several hundreds of genes and have been classified in different families according to sequence features on their carboxyl terminal domains. While these Cterminal domains are variable and are thought to be implicated in the function of the protein, the Nterminal domains are highly conserved and characterized by the presence of a LFLAK amino acid motif implicated in the translocation from the pathogen to the host cell. *A.euteiches* expresses during infection two families of CRNs, AeCRN5 and AeCRN13, both presenting a LYLALK motif responsible for the internalization of the protein inside plant cells. Both proteins are expressed during infection of *M. truncatula* roots. *In planta* expression of both proteins has revealed that AeCRN5 and AeCRN13 are targeted to the nucleus. Their expression in roots alters root architecture by inhibiting root development, while triggerring cell death in *N. benthamiana* leaves. Such observations suggest that *A. euteiches* 's CRNs are virulence proteins exerting their function through the interaction with nuclear compounds. Latest results concerning their characterization will be presented in the poster.

PS08-329

Seeing the world outside: a virus uses the host sensorial system to take cues from the environment

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Viruses rely totally on the host to achieve every step of the infection cycle. Much is known about how viruses interfere with cellular processes to put them at their use and it is clear that they intercept intracellular and intra-host communication and processes to optimise interaction with the host. Here we unprecedentedly that show viruses are also able to use the host sensorial system to very rapidly perceive and react on cues from the world outside the host, in a way disconnected from the reaction of the host itself. *Cauliflower mosaic virus* (CaMV) is transmitted from plant-to-plant by aphids, and previous work has shown that the virus-aphid interaction is not an accidental process but depends on the presence of the virus-induced Transmission Bodies (TBs) in infected cells, containing the CaMV transmissible complexes. Our results demonstrate that TBs react on the presence and feeding of the insect vector by rapidly and reversibly dispersing their contents on cortical microtubules throughout the cell. If this TB reaction is perturbed, transmission rates drop; if this reaction is artificially enhanced, transmission rates rise. This shows that CaMV intercepts the host's perception of the aphid and immediately translates it in an appropriate response that optimises its chances of acquisition, everything going back to normal standby state a few minutes later.